

The Finite Element Implementation of a K.P.P. Equation for the Simulation of Tsetse Control Measures in the Vicinity of a Game Reserve

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Abstract

An equation, strongly reminiscent of Fisher's equation, is used to model the response of tsetse populations to proposed control measures in the vicinity of a game reserve. The model assumes movement is by diffusion and that growth is logistic. This logistic growth is dependent on an historical population, in contrast to Fisher's equation which bases it on the present population. The model therefore takes into account the fact that new additions to the adult fly population, are in actual fact, the descendents of a population which existed one puparial duration ago, furthermore, that this puparial duration is temperature dependent. Artificially imposed mortality is modelled as a straight-forward, linear rate. Fisher's equation is also solved as a formality.

*The temporary imposition of a 2 % day⁻¹ mortality everywhere outside the reserve for a period of 2 years will have no lasting effect on the influence of the reserve on either the *Glossina austeni* or the *G. brevipalpis* population, although it certainly will eradicate tsetse from poor habitat, outside the reserve. A 5 km-wide barrier with a minimum mortality of 4 % day⁻¹, throughout, will succeed in completely isolating a worst-case, *G. austeni* population and its associated trypanosomiasis from the surrounding areas. A more optimistic estimate of its mobility suggests a mortality of 2 % day⁻¹ might suffice. For a given mortality, more mobile species are found to be more vulnerable to eradication than more sedentary species, while the opposite is true for containment.*

Keywords: Kolmogoroff–Petrovsky–Piscounoff; K.P.P.; Fisher's equation; tsetse; *Glossina brevipalpis*; *Glossina austeni*; trypanosomiasis; congolense; vivax.

1 Introduction

Various tsetse control measures in the vicinity of a game reserve are experimented within a simulation context. The *G. brevipalpis* and *G. austeni* populations in and around the Hluhluwe–iMfolozi Game Reserve are the object of interest. The problem posed is essentially that of the influence of a reserve of a particular size and geometry on tsetse population levels outside its confines and what can be done about it. It is based on the premise that the reserve is a problem and that it is the cause of unusually high tsetse numbers in the adjacent agricultural areas. The animals in the reserve are, moreover, considered to be a reservoir of trypanosomes and, particularly, more lethal strains.

Little is known of the tsetse species in question and so-called worst-case values must be assumed. The implementation accordingly does not correspond exactly to reality, instead, is rather simplistic and more humble than the competency of the model itself allows.

Vector Competence

The predominant infection is that of *Trypanosoma Congolense*, *T. Vivax* being prevalent to a far lesser extent. It is noteworthy that out of 900 *G. brevipalpis* teneral split into 3 equal groups and respectively allowed to feed on a different parasitaemic animal, the midgut of 4 % and the preboscis of 0 % were found to be infected (MOTLOANG, MASUMU, VAN DEN BOSSCHE, MAJIWA and LATIF [20]). The prevalence of preboscis infection for the same experiment involving *G. austeni* teneral was, in contrast, 12 % and a further 19 % were found to have an infected midgut (MOTLOANG, MASUMU, VAN DEN BOSSCHE, MAJIWA and LATIF [20]).

MOTLOANG, MASUMU, VAN DEN BOSSCHE, MAJIWA and LATIF [20] also conducted a second experiment in which they challenged each of 7 susceptible bovines with a different *G. brevipalpis* catch taken from the wild in an insect-proof facility (the combined catches totalling 468 specimens). No infection resulted. The same trial was then conducted by challenging each of 2 bovines and 1 goat with a different *G. austeni* catch taken from the wild (the combined catches totalling a mere 43 specimens only). All three challenges resulted in infection.

Both prevalence and transmission rates are therefore exceptionally high for *G. austeni* while they are virtually non-existent in the case of *G. brevipalpis*. The issue of mechanical infection by *G. brevipalpis* is currently under investigation by the same authors.

The Hluhluwe–iMfolozi Game Reserve

The Hluhluwe–iMfolozi Game Reserve is located in the southern vicinity of 28°S and 32°E, in KwaZulu–Natal, South Africa. The reserve measures some 960 km².

Inland of the coastal plain and set in the foothills of the escarpment, the temperature of the region is somewhat elevated for its latitude. The once mighty Black and White iMfolozi rivers

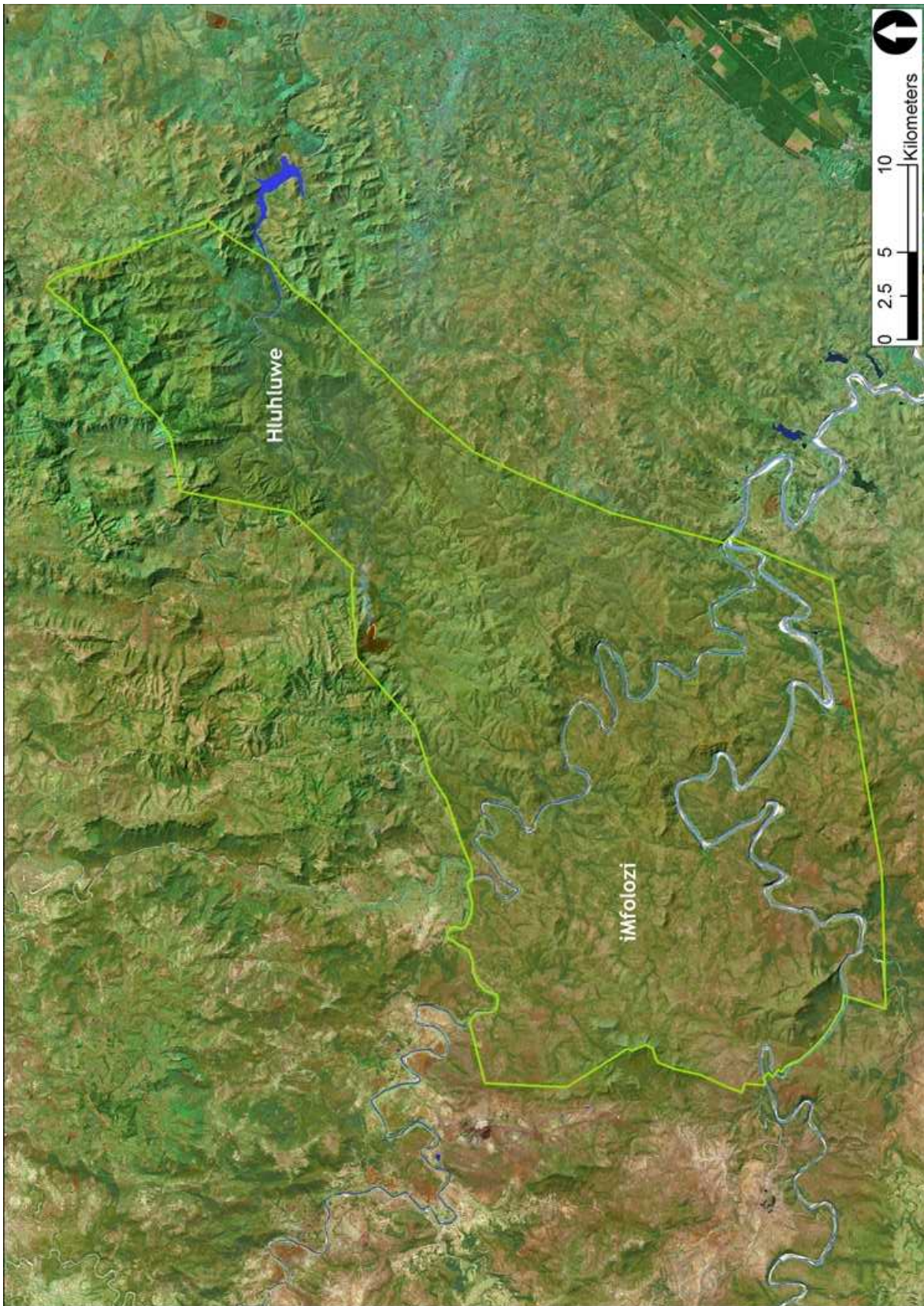


Figure 1: Satellite Image of the Hluhluwe–iMfolozi Game Reserve and its surroundings.

meander through the reserve to their confluence in the iMfolozi sector (see Figure 1), while the Hluhluwe river passes through the reserve at the Hluhluwe end (see Figure 1). It is noteworthy, with regard to both *G. brevipalpis* and *G. austeni*, that the Hluhluwe river has a flood plain within the reserve and that the backwater of the Hluhluwe Dam also extends well into it. At around this position, the reserve is approximately only 25 km from the St. Lucia estuary, a world heritage site which lies to the East.

The geomorphology of the iMfolozi lowlands is one of deep, sweeping, valleys carved out by ancient rivers, which recedes into more rugged hill-terrain. Open grasslands, savannah and woodlands characterise the iMfolozi sector. In contrast, the Hluhluwe sector gives rise to ridges as high as 540 metres above mean sea level. Precipitation at these levels is sufficient to sustain coastal, scarp forest with lush valley bushveld below. The vegetation type of the whole reserve could broadly be described as typical Zululand Lowveld, interspersed with minor Northern Zululand Sourveld. Riverine forest and thicket make the reserve the habitat of both *G. brevipalpis* and *G. austeni* and these two species, in association with large populations of buffalo and other wild animals, lead it to be something of a thorn in the side of neighbouring agriculture. Habitat outside the reserve is degraded to the extent that the boundary of the reserve is discernable in satellite images.

The Hluhluwe–iMfolozi Game Reserve has the distinction of being the oldest proclaimed game reserve in Africa. It is a lucrative tourist attraction as well as a protected area in terms of the National Environmental Management Protected Areas Act (Act no. 57 of 2003). Any tsetse control within the reserve is considered to be highly undesirable, or even out of the question from the point of view of substantial legislation and agreements cited by Ezemvelo K.Z.N. Wildlife. The reserve falls within what was once the very extensive habitat of *G. pallidipes*, a species which was completely eradicated from KwaZulu–Natal in the first half of the 20th century.

Kolmogoroff–Petrovsky–Piscounoff Equations

WILLIAMS, DRANSFIELD and BRIGHTWELL [31] originally entertained the idea of using Fisher's equation to model the distribution of tsetse populations and HARGROVE [11] devised the best implementation his circumstances permitted. The model entertained in this work is based on a very similar equation and differs mostly in the exact specification of population density in the logistic part. It belongs to a more general category of partial differential equations known as Kolmogoroff–Petrovsky–Piscounoff (K.P.P.) equations. Such partial differential equations also happen to be parabolic. In this regard, it is important to note that one cannot simply solve a parabolic, partial differential equation with a forward difference in time, nor should one use finite differences for non-rectangular geometries. The former is widely accepted as a faux pas and even in the event of circumstances which favour a correct solution, it has no credibility whatsoever. As such, the problem is ideally suited to the application of the finite element method. Fisher's equation is both parabolic and nonlinear.

Dispersal is assumed to occur by a process similar to diffusion, self-regulating growth is assumed to be logistic and a straight-forward linear rate is used to model any artificially imposed

mortality. The flies are assumed to move with some kind of Brownian motion down a diffusion gradient based on the random nature of their movement (observed by BURSELL [2] and demonstrated by ROGERS [25]). Fisher's equation is not perfectly suited to tsetse application owing to the large puparial duration which characterises the *Glossina* genus. Any growth in the present fly population has its origins in an historical fly population; one which existed one puparial duration ago. This puparial duration is temperature dependent. While Fisher's equation was largely solved for academic reasons, it offered some interesting insights.

The model itself exceeded 5000 lines of fairly extensively commented Fortran, while the mesh generator exceeded 1700 lines.

G. brevipalpis* and *G. austeni

G. brevipalpis and *G. austeni* are, in all likelihood, not the most ideal species for this type of application. Both forest species are thought to be fairly specialised and therefore habitat-specific. This observation is independently born out by the ROGERS and ROBINSON [24] study (based on FORD and KATONDO [9]'s maps) as well as the pupal water loss model in CHILDS [4]. *G. brevipalpis* would, more generally, appear to be closely associated with the riverine forest, or thicket, adjacent to drainage lines. While its pupal habitat appears to be more stringently confined than that of *G. austeni* (according to demonstrations of the pupal water loss model in CHILDS [4]), the present work will suggest *G. brevipalpis* to be more far-ranging than *G. austeni*. The latter is thought to be relatively sedentary in addition to being fickle in habitat. Both species might therefore not subscribe well to the diffusion model in venturing outside preferred habitat. ROGERS' [25] experiments with *G. fuscipes fuscipes* were in fairly uniform habitat and even then there was light-sensitive preference. Nothing appears to be known about the diffusion rates of *G. brevipalpis* and *G. austeni*. The pupal durations also subscribe worst (PARKER [22]) to the quantitative work done by PHELPS and BURROWS [23] (subsequently modified by HARGROVE [13]). One would certainly prefer to be modelling savannah species.

2 Derivation of a Model

The aim of the model is to predict how a tsetse population becomes distributed in space and how this distribution changes over time, through migration, self-regulating growth and artificially-imposed mortality. The intention is to predict a population density, $\rho(\mathbf{x}, t)$ (in which \mathbf{x} and t are space and time respectively), based on the these phenomena.

The subject of the intended model is the vector of trypanosomiasis, namely adult tsetse flies. Pupae neither migrate, nor do they (or any flies belonging to the nulliparous cohort, for that matter) form any part of the actively reproductive population. For these reasons the population density, $\rho(\mathbf{x}, t)$, is defined not to include pupae. While it is tempting to also exclude any flies belonging to the nulliparous cohorts from a reproductive point of view, such flies are mobile and subject to the external, artificial mortality to be imposed; indeed, the subject of

this investigation. While the correspondence of the reproductive population to the mobile and vulnerable population is not perfect, it is suitably close.

2.1 Describing the Three Dynamics of Interest

How might one model the population change brought about through migration, self-regulating growth and artificially-imposed mortality? If all three effects can be regarded as being mutually independent of one another, they can be considered in isolation.

2.1.1 Migration

BURSELL [2] put forward the theory that the movement of tsetse was of a random nature, not unlike Brownian motion, and ROGERS [25] proved these assertions quantitatively. If one can conceive of a gas as a continuum, it is only slightly more abstract to conceive of a fly population as a continuum.

Consider the hypothetical scenario of a mobile population in the absence of either reproduction or mortality. Biomass should therefore be conserved and the standard continuum-mechanical result for mass conservation pertains. It can be manipulated to give a result not unlike the Reynolds transport theorem and Fick's first law applied. (A full exposition is provided in the addendum.) The resulting rate for the effect of migration in isolation is

$$\frac{\partial \rho}{\partial t} = \lambda \operatorname{div} \nabla \rho$$

in which λ is the diffusion coefficient.

2.1.2 Self-Regulating Growth

The logistic model needs little introduction to a biological audience. The population is assumed to grow at a rate which is some proportion, r , of some parent population ρ^* . Reason dictates that this growth rate must necessarily also be constrained by the carrying capacity, K , of the environment. The simplest and most obvious construct which will accomplish this is a factor $1 - \frac{\rho}{K}$. Such a factor allows the growth rate to drop off linearly as the population level rises. The resulting rate for self-regulating growth in isolation could therefore be modelled as

$$\frac{\partial \rho}{\partial t} = r \rho^* \left(1 - \frac{\rho}{K} \right).$$

Larval production is clearly dependent on the population which existed one puparial duration ago in the tsetse context. What about population density in the second factor of the logistic term; the one limiting the growth rate? The pertinent population is not as obvious in this instance. Combined pupal and teneral mortality is an order of magnitude higher than adult

mortality (HARGROVE [13]) and VALE [29] seems to think that parasitism alone accounts for between 40% to 60% of the overall pupal mortality, under usual circumstances in a favourable environment. Quantitative work linking predation and parasitism to the density at pupal sites has been carried out by ROGERS and RANDOLPH [26]. That work could therefore be taken to recommend a logistic term based entirely on an historical population density, that which existed at the time of larval deposition and parturition.

Can such a model be reconciled with the other, remaining causes of pupal mortality? Although fat loss¹ and water loss¹ are determined by external variables of temperature and humidity, indirect dependence on population density is possible in the event of a shortage of available breeding sites. Spatial variation of temperature and humidity is incorporated, up to a point, in the carrying capacity and growth rate of each environment. It is important to note, however, that the effects of any temporal variation in the growth rate, r , are beyond the scope of the standard logistic model, although it does allow for a time-dependent carrying capacity, K . In this particular model pupal metabolism and development are temperature-dependent, therefore time-dependent. Including this dependence allows the origins of presently ecdoding pupae to be traced to their parent population.

The fact that an historical population level was responsible for both larval production and subsequent, density-dependent mortality is taken into account in this particular model. Both larval production and subsequent natural mortality are dependent on the historic population level, that which existed one puparial duration ago. The population density at the time of larval deposition was

$$\rho^*(\mathbf{x}, t) \equiv \rho(\mathbf{x}, t - \bar{\tau}),$$

in which $\bar{\tau}$ is the relevant puparial duration.

The correspondence of the reproductive population to the mobile and vulnerable population is not perfect, however, this can readily be taken into account by assuming a fixed age profile for the growth rate.

ASSUMPTION 1 *The age profile of the population is fixed.*

How reasonable is this assumption? One consequence is that the imposition of any artificial, adult-selective mortality gives rise to a damped logistic response from the model above $K/2$ and an over-reactive one below $K/2$. In reality, a smaller proportion of reproductive adults than the model supposes, exists. The greater part of the logistic curve may be approximated as linear, as can the entire curve, locally. No problems should be expected for reasonable use. “Reasonable use” does not, however, include scenarios such as aerial spraying.

At some point what is presently being couched in terms of a slightly extended first interlarval period becomes the phase entrainment of the reproductive population. Tsetse reproduction is not continuous. Instead, it occurs at discrete intervals dictated by the first and subsequent

¹Teneral mortality from both fat loss and water loss is thought to be high and is often the cumulative effect of temperature and humidity conditions which prevailed during the pupal phase.

interlarval periods. Problems could therefore be anticipated, should the age profile of the general population become significantly altered by any artificially imposed, adult-selective carnage (e.g aerial spraying) and the subsequent population not be allowed sufficient time to re-equilibrate. Pupae, about to emerge, would not be susceptible to the aforementioned mortality. Shortly afterwards, a dramatic change in the age profile of the population is induced by the addition of abnormally large proportions of young flies of an unreplicative age. The model fails to recognize the abnormally youthful population as such and assumes it to be reproductive. What in reality should be a reproductive ‘shadow’, a dearth of reproductive flies (which the model does not recognize), leads to an over-estimated eclosion exactly one puparial duration later. These fictitious, newly-emerged flies, nonetheless, assist in compensating for what, in reality, is yet another skewed age profile; this time a surplus of now-mature and reproductive flies. Their maturity would otherwise lead to an underestimated eclosion, yet another puparial duration later, and so on. Fortunately the tsetse fly is a K -strategist and, given enough time, the model is expected to re-equilibrate. It would obviously not recover from a sequence of such events in close succession. This limitation is negligible in comparison to one presently to be discussed in connection with a model based on Fisher’s equation.

2.1.3 Artificially Imposed Mortality

Suppose that the effect of targets, pour-ons etc. is to cause the population density to decline according to $\rho\delta t$, where δ is independent of the population density. Then the resulting rate for artificially imposed mortality in isolation is

$$\frac{\partial \rho}{\partial t} = -\delta \rho.$$

2.2 A Governing Equation

The combined effect of all three phenomena is additive and a model can therefore be based on the following equation. Two alternatives arise based on the exact specification of the parent population density, $\rho^*(\mathbf{x}, t)$. In the equation

$$\frac{\partial \rho(\mathbf{x}, t)}{\partial t} = \lambda \operatorname{div} \nabla \rho(\mathbf{x}, t) + r \rho^*(\mathbf{x}, t) \left(1 - \frac{\rho(\mathbf{x}, t)}{K} \right) - \delta \rho(\mathbf{x}, t), \quad (1)$$

$\rho(\mathbf{x}, t)$ is otherwise the current population density (in which \mathbf{x} and t are space and time respectively), λ is an diffusion rate, r is the population growth rate, K is the carrying capacity of the environment and δ is an artificially imposed mortality. The quantity $\rho^*(\mathbf{x}, t)$ is either an historical population density or the current population density, depending on the model preferred.

REMARK: Notice that in the special case of $\rho^*(\mathbf{x}, t) = \rho(\mathbf{x}, t)$ and $\delta = 0$, equation 1 becomes immediately recognizable as Fisher’s equation in its classical form. It is otherwise part of a more general and widely inclusive family, known as Kolmogoroff–Petrovsky–Piscounoff equations.

2.2.1 Fisher's Equation

As WILLIAMS, DRANSFIELD and BRIGHTWELL [31] rightly observe, equation 1 can, in the special case of $\rho^*(\mathbf{x}, t) = \rho(\mathbf{x}, t)$, be manipulated to a Fisher's equation in which there are new, modified carrying capacities and growth rates,

$$K' = K \left(1 - \frac{\delta}{r}\right) \quad \text{and} \quad r' = r \left(1 - \frac{\delta}{r}\right),$$

respectively. A logistic term dependent on the present population density is, however, something known to be incorrect. The larval deposition responsible for growth in the present population took place a significant time previously. In the Fisher's-equation model the existence of the pupal phase is denied, alternatively, pupae are assumed to migrate and reproduce.

Why use an equation which is less appropriate? Firstly, it would be interesting to know if there is any difference between the K.P.P.-model results and those arising from the unquestioning application of Fisher's equation; given the long periods to equilibrate that this particular application will allow. Secondly, convergence with little or no iteration will indicate the steady state. Thirdly, there is a certain amount of academic interest as the mathematics is more challenging. If the numerical techniques employed are powerful enough to solve a nonlinear Fisher's equation, they will, logically, solve an equation with any other variants of the logistic term contemplated. (One might later wish to model another vector, like *culicoides*.) Fourthly, a point already mentioned is that if the age profile in the model of interest is altered in such a way that it contains a significantly higher than usual proportion of flies of a nulliparous age, then the growth rate (which is based on a fixed age profile) in the model may no longer be appropriate. Just how reasonable is the assumption of a fixed age profile? Fisher's equation makes a far worse assumption and a comparison between results might indicate the extent of the problem.

3 Scaling

Suppose that T is (only for the present) a unit of time, X is a unit of length and η is a characteristic population density. The scaled variables are then

$$\mathbf{x} = \bar{\mathbf{x}}X, \quad t = \bar{t}T \quad \text{and} \quad \rho = \bar{\rho}\eta \quad (\text{including } K = \bar{K}\eta).$$

The operators become

$$\frac{\partial}{\partial \mathbf{x}} = \frac{1}{X} \frac{\partial}{\partial \bar{\mathbf{x}}} \Rightarrow \operatorname{div} \nabla \rho = \frac{\eta}{X^2} \operatorname{div} \bar{\nabla} \bar{\rho}$$

and

$$\frac{\partial}{\partial t} = \frac{1}{T} \frac{\partial}{\partial \bar{t}} \Rightarrow \frac{\partial \rho}{\partial t} = \frac{\eta}{T} \frac{\partial \bar{\rho}}{\partial \bar{t}}$$

in terms of the scaled variables. Thus

$$\frac{\eta}{T} \frac{\partial \bar{\rho}}{\partial \bar{t}} = \frac{\eta}{X^2} \lambda \operatorname{div} \bar{\nabla} \bar{\rho} + r\eta \bar{\rho}^* \left(1 - \frac{\bar{\rho}}{\bar{K}}\right) - \delta\eta \bar{\rho}.$$

All of this suggests using $T = \frac{X^2}{\lambda}$ and $X = \sqrt{\frac{\lambda}{r}}$ so that the above equation becomes

$$\frac{\partial \bar{\rho}}{\partial \bar{t}} = \bar{\text{div}} \bar{\nabla} \bar{\rho} + \bar{\rho}^* \left(1 - \frac{\bar{\rho}^*}{\bar{K}} \right) - \frac{\delta}{r} \bar{\rho}.$$

If the mesh is in units of kilometres, for example, then it must be converted by dividing through by $\sqrt{\frac{\lambda}{r}}$ kilometres. One would imagine the desire to multiply positions by $\sqrt{\frac{\lambda}{r}}$ when outputting the solution, thereby returning to kilometres.

Complication: If one intends including any environmental variation in the rates of diffusion and growth, the scaled equation will entail different and therefore irreconcilable time steps.

$$\frac{\partial \bar{\rho}}{\partial \bar{t}} = \frac{\lambda}{\lambda_{scale}} \bar{\text{div}} \bar{\nabla} \bar{\rho} + \frac{r}{r_{scale}} \bar{\rho}^* \left(1 - \frac{\bar{\rho}^*}{\bar{K}} \right) - \frac{\delta}{r_{scale}} \bar{\rho}. \quad (2)$$

The above equation allows a time discretisation which conforms.

4 Variational Formulation

A variational formulation of equation 2 is obtained in the usual fashion: Premultiplying the primitive variable equation by an arbitrary function, w , and integrating over the domain, Ω , to obtain

$$\int_{\Omega} w \frac{\partial \rho}{\partial t} d\Omega = \frac{\lambda}{\lambda_{scale}} \int_{\Omega} w \text{div} \nabla \rho d\Omega + \frac{r}{r_{scale}} \int_{\Omega} w \rho^* \left(1 - \frac{\rho^*}{K} \right) d\Omega - \frac{\delta}{r_{scale}} \int_{\Omega} w \rho d\Omega \quad (3)$$

The approximation-wise cumbersome second derivatives appearing in this equation can also be avoided in the usual fashion. The term which contains the divergence of $\nabla \rho$ can be regarded as one part of a differentiated product and the divergence theorem applied so that

$$\int_{\Omega} w \frac{\partial \rho_{,i}}{\partial x_i} d\Omega = \int_{\Gamma} w \rho_{,i} n_i d\Gamma - \int_{\Omega} w_{,i} \rho_{,i} d\Omega,$$

in which \mathbf{n} is the outward unit normal and Γ is the domain boundary. Of the two terms resulting, one is a boundary integral. The integrand obviously vanishes for a von Neumann, ' $\mathbf{n} \cdot \nabla \rho = 0$ '-type boundary condition. The arbitrary vector of the formulation can otherwise be assigned a value of zero on boundaries where boundary conditions are Dirichlet (and variational formulation is consequently not required). The boundary integral is zero along such boundaries.

5 Discretisation

A backward difference is used for the temporal discretisation, while the finite element method is used for the spatial discretisation. The solution at time t is accordingly assumed to be a linear

combination of shape functions, $\psi(\mathbf{x})$. That is,

$$\rho(\mathbf{x})|_t = \sum_{i=1}^N c_i \psi_i(\mathbf{x}),$$

where the c_i are the constants of the finite element approximation (the nodal solutions) and N is the total number of nodes. The problem on each element is calculated in terms of a standard, master element coordinate system, $\{\xi\}$.

5.1 Finite Element Construction

The approximate equation, to be solved for the nodal population densities, P_j^e (pertaining to element e), are consequently

$$\begin{aligned} \mathbf{A}_{e=1}^E \left\{ \frac{1}{\Delta t} \int_{\hat{\Omega}} \phi_i \phi_j J^e d\hat{\Omega} + \frac{\lambda}{\lambda_{\text{scale}}} \int_{\hat{\Omega}} \frac{\partial \phi_i}{\partial x_k} \frac{\partial \phi_j}{\partial x_k} J^e d\hat{\Omega} + \frac{\delta}{r_{\text{scale}}} \int_{\hat{\Omega}} \phi_i \phi_j J^e d\hat{\Omega} \right\} \mathbf{A}_{e=1}^E P_j^e \\ = \mathbf{A}_{e=1}^E \left\{ \frac{1}{\Delta t} \int_{\hat{\Omega}} \phi_i \phi_m J^e d\hat{\Omega} P_m^e|_{t-\Delta t} + \frac{r}{r_{\text{scale}}} \int_{\hat{\Omega}} \phi_i \phi_n J^e d\hat{\Omega} P_n^e|_{t-\bar{\tau}} \right. \\ \left. - \frac{r}{r_{\text{scale}}} \int_{\hat{\Omega}} \phi_i \phi_l \frac{\phi_j}{K} J^e d\hat{\Omega} P_l^e|_{t-\bar{\tau}} P_j^e|_{t-\bar{\tau}} \right\}, \end{aligned} \quad (4)$$

in which \mathbf{A} is the element assembly operator, E is the total number of elements, e , into which the domain has been subdivided, $\hat{\Omega}$ is the master element domain, Δt is the length of the time step, the $\phi_i(\xi)$ are the basis functions,

$$\frac{\partial \phi_i}{\partial x_j}(\xi) = \frac{\partial \phi_i}{\partial \xi_k} \frac{\xi_k}{\partial x_j}, \quad J^e = \left| \frac{\partial \mathbf{x}}{\partial \boldsymbol{\xi}} \right| \text{ for element } e,$$

λ is the rate of diffusion, λ_{scale} is a diffusion–rate scale, r is the population growth rate, r_{scale} is a population growth–rate scale, K is the carrying capacity of the environment and δ is an artificially imposed mortality. $P_n^e|_{t-\bar{\tau}}$ denotes the solution at the time of larval deposition (that which led to the present eclosion), $\bar{\tau}$ being the relevant average of puparial durations.

Fisher's Equation

The analogous finite element implementation for Fisher's equation is

$$\begin{aligned} \mathbf{A}_{e=1}^E \left\{ \frac{1}{\Delta t} \int_{\hat{\Omega}} \phi_i \phi_j J^e d\hat{\Omega} + \frac{\lambda}{\lambda_{\text{scale}}} \int_{\hat{\Omega}} \frac{\partial \phi_i}{\partial x_k} \frac{\partial \phi_j}{\partial x_k} J^e d\hat{\Omega} - \frac{r}{r_{\text{scale}}} \int_{\hat{\Omega}} \phi_i \phi_j J^e d\hat{\Omega} \right. \\ \left. + \frac{r}{r_{\text{scale}}} \int_{\hat{\Omega}} \phi_i \phi_j \frac{\phi_l}{K} J^e d\hat{\Omega} P_l^e \text{ linearisation} + \frac{\delta}{r_{\text{scale}}} \int_{\hat{\Omega}} \phi_i \phi_j J^e d\hat{\Omega} \right\} \mathbf{A}_{e=1}^E P_j^e \\ = \mathbf{A}_{e=1}^E \left\{ \frac{1}{\Delta t} \int_{\hat{\Omega}} \phi_i \phi_m J^e d\hat{\Omega} P_m^e|_{t-\Delta t} \right\}, \end{aligned} \quad (5)$$

in which

$$\mathbf{P}^e \text{ linearisation} = 2\mathbf{P}^e|_t - \mathbf{P}^e|_{t-\Delta t},$$

the second order accurate linearisation originally used in CHILDS [3].

6 The Relevant Parental Population

Determining the puparial duration leading to the present eclosion is a minor problem in its own right. It is known that at a given temperature, T , the puparial duration, in days, can be calculated according to the formula

$$\tau(T) = \frac{1 + e^{a+bT}}{\kappa}, \quad (6)$$

(PHELPS AND BURROWS [23]). For females, $\kappa = 0.057 \pm 0.001$, $a = 5.5 \pm 0.2$ and $b = -0.25 \pm 0.01$. For males, $\kappa = 0.053 \pm 0.001$, $a = 5.3 \pm 0.2$ and $b = -0.24 \pm 0.01$ (HARGROVE [13]). The puparial durations of all species, with the exception of *G. brevipalpis*, are thought to lie within 10% of the value predicted by this formula (PARKER [22]). *G. brevipalpis* takes a little longer. The shortest puparial duration is that of *G. austeni*.

If $\bar{\tau}$ is the relevant average of puparial durations (which is, of course, dependent on itself) then

$$\begin{aligned} \bar{\tau} \equiv \frac{1}{\bar{\tau}} & \left[\left[t - \text{floor}\{t\} \right] \tau(T_{\text{ceil}\{t\}}) + \sum_{i=\text{floor}\{t\}}^{\text{ceil}\{t-\tau+1\}} \tau(T_i) \right. \\ & \left. + \left[\text{ceil}\{t-\tau\} - (t-\tau) \right] \tau(T_{\text{ceil}\{t-\tau\}}) \right], \end{aligned}$$

in which $\tau(T)$ is given by the formula (6) and T is the mean daily temperature on the day indicated by the subscript. Newton's method is used in solving the above equation. The relevant parental population at the time $t - \bar{\tau}$ is a weighted average of the nearest two solutions since a backward difference was used for the temporal discretisation.

7 Implementation in the Context of the Reserve and its Surroundings

A suitable finite element mesh had to be generated and pertinent carrying capacities, growth rates and diffusion coefficients associated with it. Little is known of the tsetse species in question and so-called worst-case values must be assumed. The implementation accordingly does not correspond exactly to reality, instead, is rather simplistic and more humble than the competency of the model itself allows.

The carrying capacities were also the initial, start-up values assumed, with the exception of the northern and western boundaries, where the population was assumed to be 0%. A constant

supply of flies maintaining the eastern and southern boundaries of the region at a 5% level was assumed (regardless of the controls experimented with). The model also requires daily temperature data.

The lack of statistically significant data and the simplicity of an initial case study were deemed to mitigate the following assumption.

ASSUMPTION 2 *There are no seasonal, or temporal, changes in the environment.*

In other words the model assumes that any non-density-dependent causes of pupal mortality are constant in time, although the potential to vary carrying capacity does exist in the model; both K and r are constant in time. This is not to say that the environment and climate do not vary spatially. Spatial variation of the environment is incorporated into the parameters of growth rate and carrying capacity. Of course in reality there are seasonal changes, however, one would hope that the forest environment would be more insulated than most. Pupal survival for *G. brevipalpis* and *G. austeni* is more especially sensitive to environmental conditions (CHILDS [4]); the environmental conditions which are incorporated into the parameters of growth rate and carrying capacity and which are, in this application, assumed not to vary in time.

7.1 The Finite Element Mesh

Nine-noded quadrilateral elements and the associated Q_2 element basis were used. A program to generate the mesh was written based on CHILDS and REDDY [5].

7.2 The Carrying Capacity, K

The *Glossina* genus is a K -strategist and carrying capacities are therefore a deciding factor in the implementation. The problem posed is, fundamentally, that of the influence of a reserve of a particular size and geometry on tsetse population levels outside its confines, a reserve which is a reservoir of more lethal strains and higher levels of trypanosomiasis, and what can be done about it. It was with this in mind that the decision was taken to model the carrying capacity of the region, rather simplistically, as in Table 1 and Figure 3. *G. brevipalpis* data (Figure 4) are indeed suggestive of such a reality, however, the *G. austeni* data (Figure 7) do not correspond as well to such a simplistic distribution.

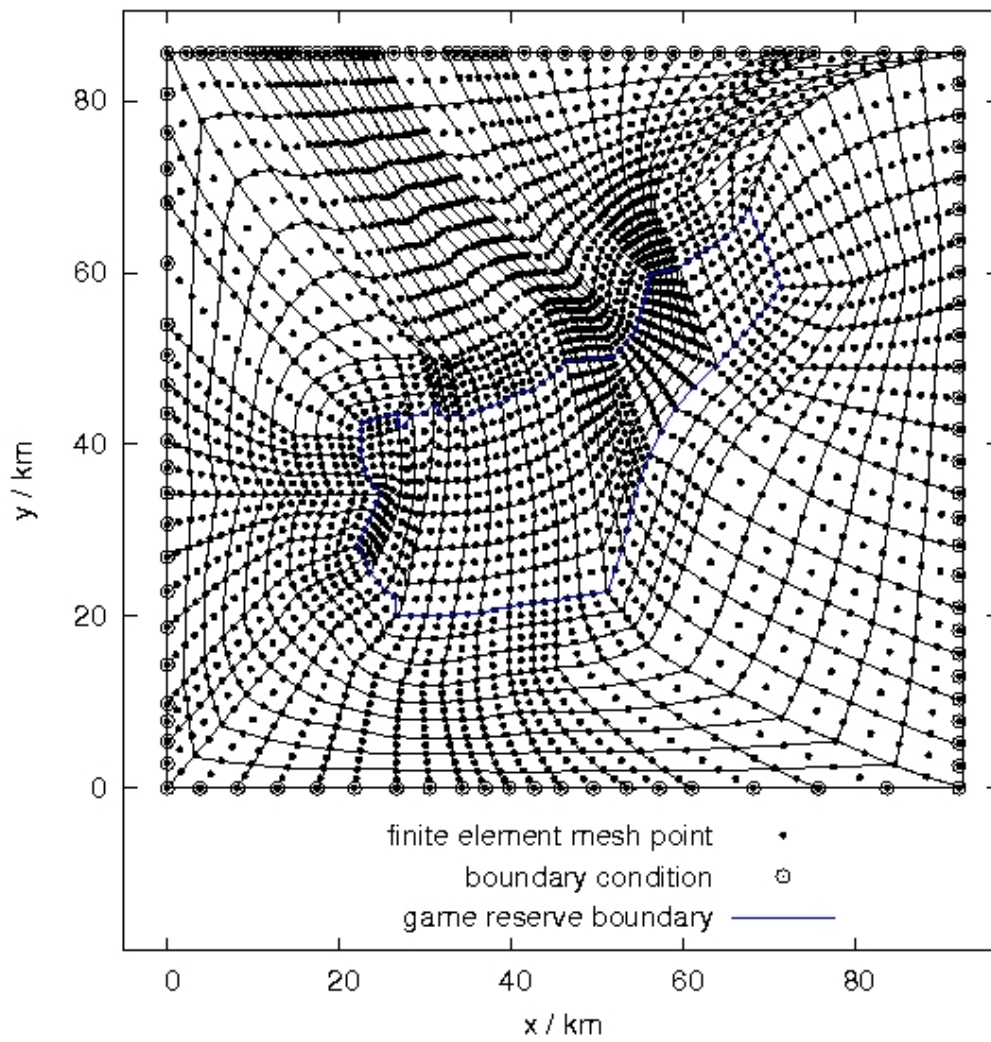


Figure 2: The finite element mesh.

$\approx d / \text{km}$	$d \leq 0$	$0 < d \leq 2.5$	$2.5 < d \leq 5$	$d > 5$
$K / \%$	100	20	10	5

Table 1: The carrying capacity, K , designated according to the approximate distance, d , from the reserve boundary.

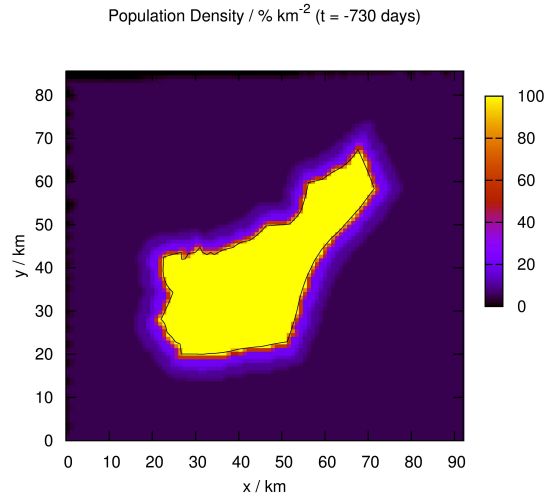


Figure 3: Start-up values and carrying capacity (with the exception of the northern and western boundaries).

In reality, the South-Western, iMfolozi section of the reserve is hotter and drier. That environment is consequently not as hospitable to *G. brevipalpis* and *G. austeni* as the lush, North-Eastern, Hluhluwe side. In reality, some suitable habitat exists outside the confines of the reserve. In reality, tsetse numbers in the East are supplemented by St. Lucia and the coastal area below it, whereas populations in the North are sometimes limited by higher ground.

7.3 The Maximum Growth Rate, r

Growth rates were assigned in such a way as to correspond to these self-same zones. At Hluhluwe–iMfolozi mean annual temperatures, each female would produce 4 pupae based on GLASGOW [10]’s 49-day, average, adult life-span. The population would therefore grow by $2.4\% \text{ day}^{-1}$ in the absence of any early mortality, assuming an equal ratio of the sexes. Of course, in reality there is massive pupal and teneral mortality. In reality, the growth rate is probably

closer to $0.85\% \text{ day}^{-1}$ (HARGROVE [13]). A worst-case value of $1.1\% \text{ day}^{-1}$ in good habitat was used for the purposes of this investigation.

$\approx d / \text{km}$	$d \leq 0$	$0 < d \leq 2.5$	$2.5 < d \leq 5$	$d > 5$
$r / \% \text{ day}^{-1}$	1.1	0.5	0.2	0.1

Table 2: The growth rate, r , designated according to the approximate distance, d , from the reserve boundary.

7.4 The Diffusion Coefficient, λ

No information would appear to be available on the diffusion rates of either *G. brevipalpis* or *G. austeni*, whatsoever. The most comprehensive set of measurements are probably those for *G. morsitans* recorded by JACKSON [18] (reported in ROGERS [25]). The rate at which *G. morsitans* dispersed in *G. swynnertoni* habitat was found to be a constant $0.153 \text{ km}^2 \text{ day}^{-1}$ if the initial stages of the experiment are omitted. Other work, mostly by the same author (also summarised in ROGERS [25]), suggests *G. morsitans* was possibly uncomfortable in *G. swynnertoni* habitat. The very low end of the *G. morsitans* range would appear to be about $0.04 \text{ km}^2 \text{ day}^{-1}$. The point is that different habitats can have different coefficients, as do different species, and one would imagine temperature plays a role.

G. brevipalpis and *G. austeni* are profoundly different species to *G. morsitans*, in both size and habitat. *G. morsitans* is of an intermediate size, while *G. brevipalpis* is one of the largest flies known. *G. austeni* is the smallest of the tsetse flies. *G. brevipalpis* and *G. austeni* are both forest-dwelling, whereas *G. morsitans* is a savannah species. Under such circumstances one is required to be more resourceful. The premise of this work is that the reserve is a problem, that it is the cause of unusually high tsetse numbers in the adjacent agricultural areas. Indeed, the difference in habitat which is visibly discernable in satellite images and the tsetse risk map, Figure 4, certainly are suggestive of a reserve with a zone of influence in the case of a very habitat-specific *G. brevipalpis*. The distribution of *G. austeni* (Figure 7) is less well understood. In the case of *G. austeni* it is arguable whether a likely zone of influence can be detected. (There appear to be areas of good *G. austeni* habitat outside the reserve.) An haven with a zone of influence is nevertheless a premise on which to proceed and one might speculate a smaller range i.e. a lower ‘diffusion’ rate, as a point of departure, due to the small size of this fly.

One strategy would be to proceed by trial and error with different values of the coefficient until matching zones of influence to those evidenced by Figure 4 and Figure 7 are produced. This is, in fact, exactly what was resorted to. The value of the diffusion coefficient was either halved, or doubled, until a suitable zone of influence was generated. The match was obviously not

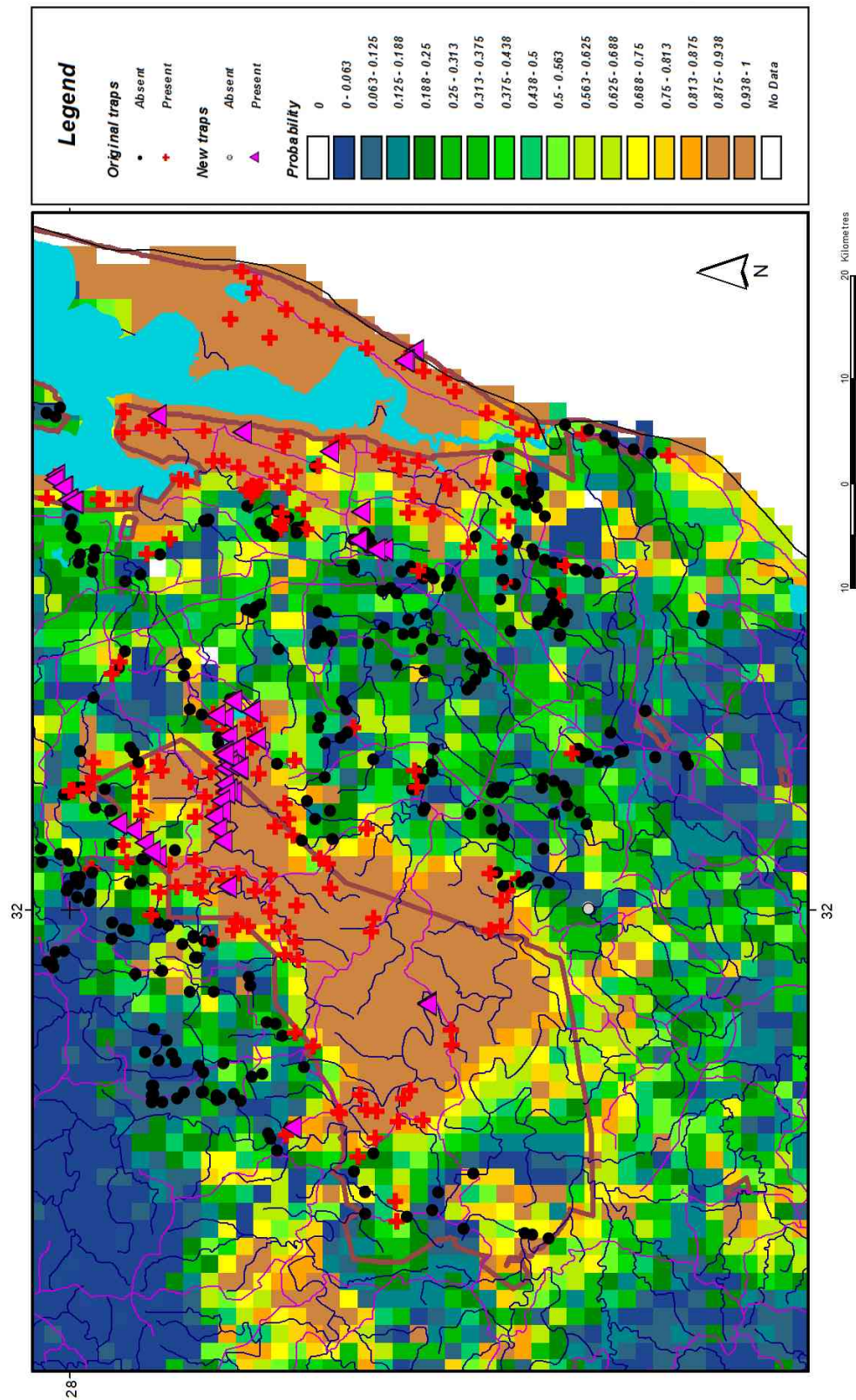


Figure 4: *G. brevipalpis* risk. The large concentration of pink triangles coincides with the position of the Hluhluwe Dam and its backwater. The minor habitat to the NW of the reserve and the diagonal of red crosses leading down from it, through the reserve, are associated with the Blue Mfolozi River (See [Hluhluwe area \[17\]](#)).

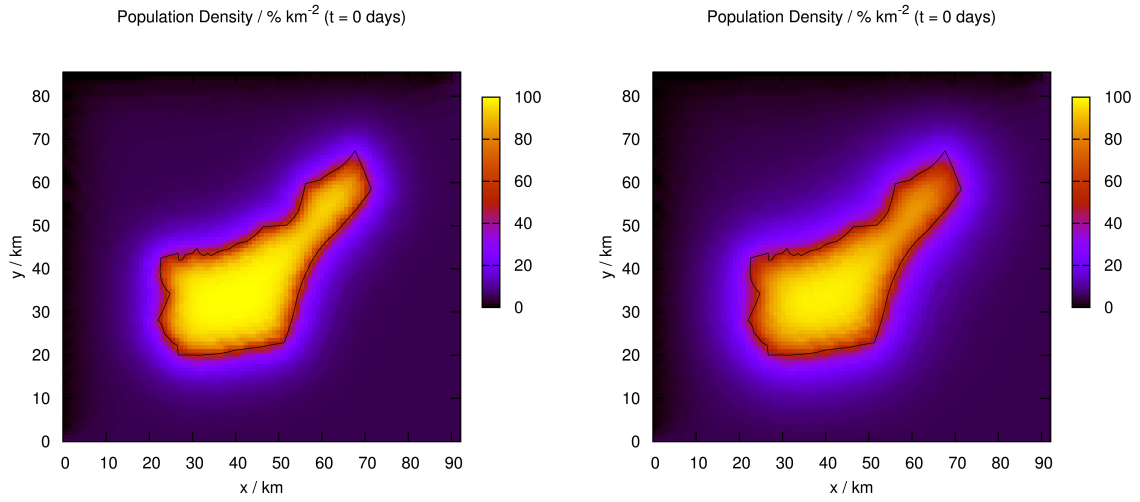


Figure 5: Computed steady states for $\lambda = 0.04 \text{ km}^2 \text{ day}^{-1}$, at left, and $\lambda = 0.08 \text{ km}^2 \text{ day}^{-1}$, at right, after 2 years.

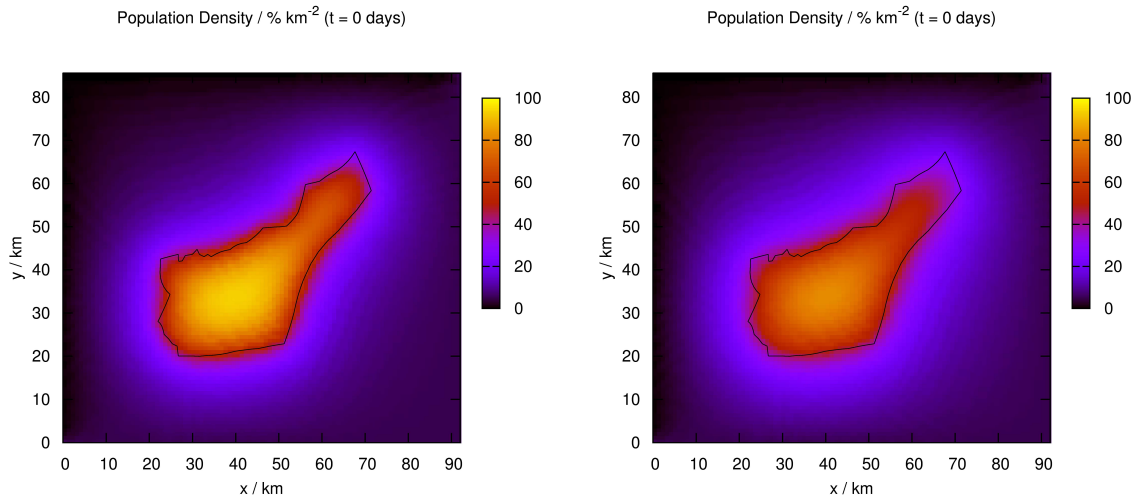


Figure 6: Computed steady states for $\lambda = 0.16 \text{ km}^2 \text{ day}^{-1}$, at left, and $\lambda = 0.32 \text{ km}^2 \text{ day}^{-1}$, at right, after 2 years. Notice that the boundary conditions are very close and starting to effect the computed zone of influence at these high diffusion rates.

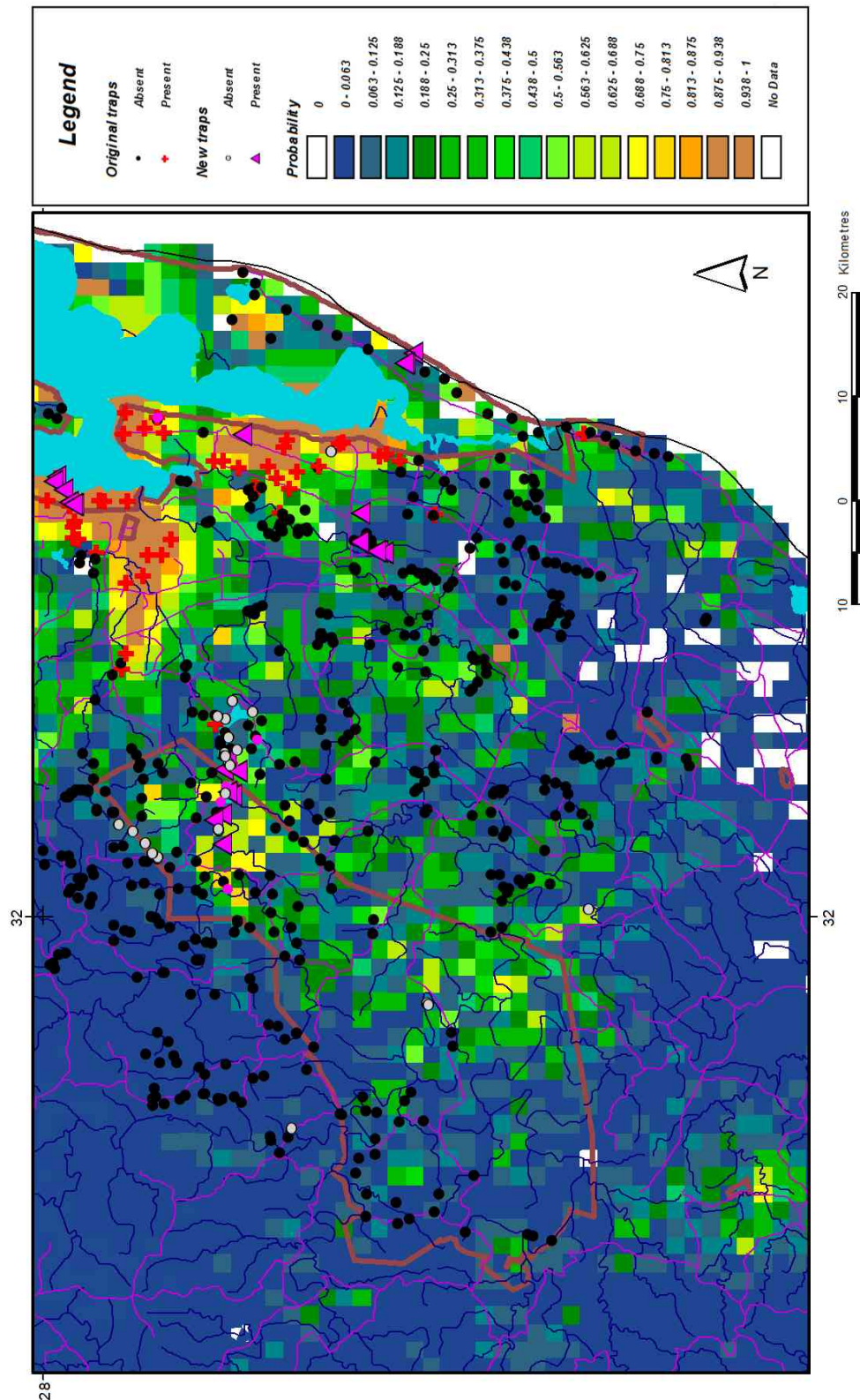


Figure 7: *G. austeni* risk. The large concentration of pink triangles indicates the position of the Hluhluwe Dam and its backwater. The minor habitat evident in the South of the reserve is in the vicinity of the confluence of the Black and White iMfolozi Rivers. (Source: HENDRICKX [17])

perfect due to higher ground to the North, suitable habitat outside the reserve (which was not modelled) and supplementation from the St. Lucia populations. With hindsight, a mesh which included a bigger area would have been preferred. The crude technique suggested some very acceptable values.

The diffusion rate of *G. austeni* is probably $0.04 \text{ km}^2\text{day}^{-1}$, that of a very comfortable *G. morsitans* population. Intuitively, this makes sense because of the small size of the species. At worst, one might speculate that it could reach $0.08 \text{ km}^2\text{day}^{-1}$. The diffusion coefficient of *G. brevipalpis*, however, came as something of a surprise for a forest-dwelling species. At somewhere between $0.16 \text{ km}^2\text{day}^{-1\dagger}$ and, very possibly as high as $0.32 \text{ km}^2\text{day}^{-1}$ (an order of magnitude larger than *G. austeni*) it approaches ROGERS [25]’s observations of *G. fuscipes fuscipes*, a fly of similar habitat, though smaller size.

7.5 The Temperature, T

The South African Meteorological Services quote a mean annual temperature of 22.1°C for Mpila, inside the reserve (based on data collected during the 1980’s and 1990’s). This is consistent with the data of SCHULZE and MAHARAJ [27], who define the overall area as being of a temperature greater than 22°C . The Mpila value is further corroborated by ARC-ISCW automatic weather stations situated between Mtunzini and Pongola (operational since 2004). They suggest an average daily temperature of 22°C , according to the Department of Agriculture.

8 Results

The results of the simulations are intended to address the following questions:

1. What are the likely, worst-case diffusion rates of *G. brevipalpis* and *G. austeni*?
2. What will the long-term effect of the temporary, 2-year use of pour-ons in the surrounding areas be?
3. Can the influence of the Hluhluwe–iMfolozi Game Reserve on surrounding tsetse population levels be negated? If so, what measures will this require?
4. Can the tsetse populations of the Hluhluwe–iMfolozi Game Reserve and their associated trypanosomiasis be completely isolated from the surrounding areas? If so, what measures will this require?
5. What is a practical barrier width?
6. Can the populations within the reserve be ‘pumped out’ to extinction from outside the reserve; failing that, down to the 20% level?

[†]that of an uncomfortable *G. morsitans* population.

7. In what way are diffusion rates relevant to containment, eradication and any subsequent rebound?

For each simulation the model was first run for two years to allow it to settle down to a steady-state. Two years was deemed more than adequate time for the model to equilibrate. This was decided on the basis of a visual inspection of the values (which were no longer changing) as well as the marked drop in the number of iterations required to solve the alternative model, based on Fisher's equation. The model was then run for another two years¹ with various controls in place, all of which were modelled at a 2 % day⁻¹ mortality to start with. The tsetse population was then allowed to rebound for a further two years, with or without controls still in place.

Barriers with a width greater than 4 km were not experimented with, even though they might be more optimal in terms of the required number of targets. This is since they were deemed to be a self-defeating waste of land.

8.1 A 2.5 km-wide Barrier Surrounding the Reserve (with a 2 % day⁻¹ Mortality Throughout)

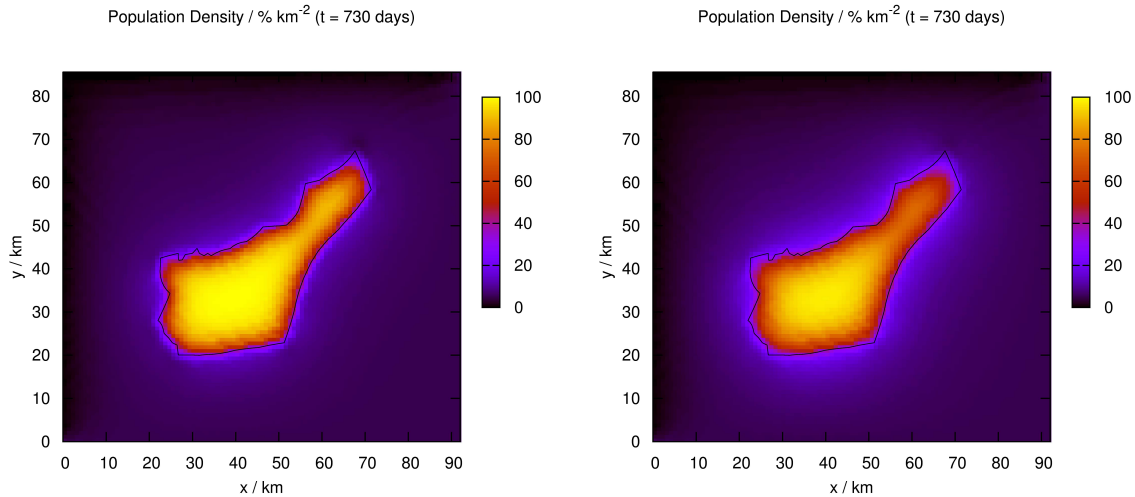


Figure 8: The results of an approximately 2.5 km-wide barrier after 2 years. At left, $\lambda = 0.04 \text{ km}^2 \text{ day}^{-1}$. At right, $\lambda = 0.08 \text{ km}^2 \text{ day}^{-1}$.

¹Deltamethrin pour-ons can not safely be used on cattle for any longer than two years without compromising their resistance to tick-bourne diseases, consequently an enzootic condition

8.2 A 5 km-wide Barrier Surrounding the Reserve (with a 2 % day⁻¹ Mortality Throughout)

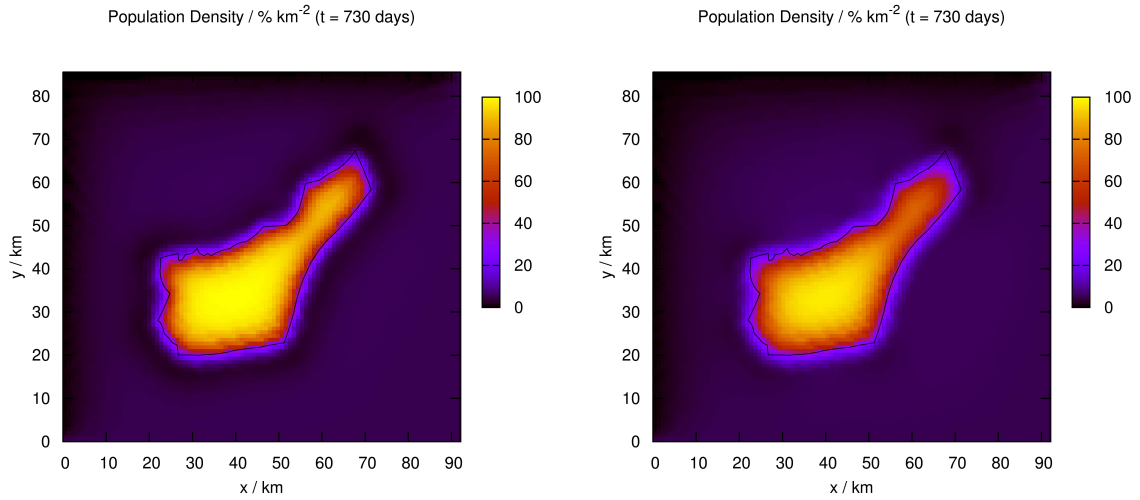


Figure 9: The results of an approximately 5 km-wide barrier after 2 years. At left, $\lambda = 0.04 \text{ km}^2 \text{ day}^{-1}$. At right, $\lambda = 0.08 \text{ km}^2 \text{ day}^{-1}$.

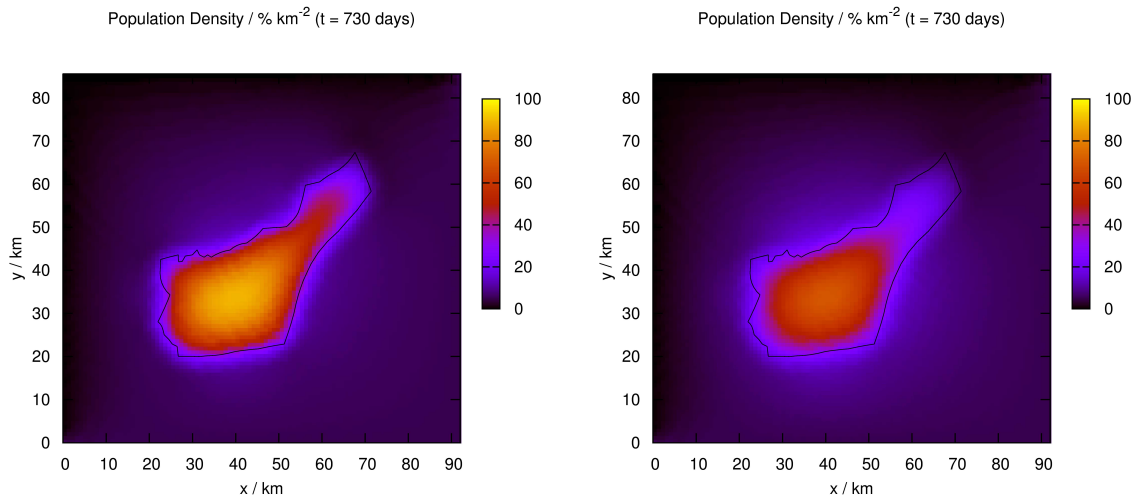


Figure 10: The results of an approximately 5 km-wide barrier after 2 years. At left, $\lambda = 0.16 \text{ km}^2 \text{ day}^{-1}$. At right, $\lambda = 0.32 \text{ km}^2 \text{ day}^{-1}$.

8.3 The Quest for an Impenetrable, 5 km–wide, Surrounding Barrier

The influence of the reserve on surrounding tsetse populations is negated in both scenarios on the left, however, flies with higher levels and more lethal strains of trypanosomiasis are still able to commute. In both right–hand side scenarios the barrier zone is completely vacant, which means no flies leave the barrier, which, in turn, means no flies ever cross it.

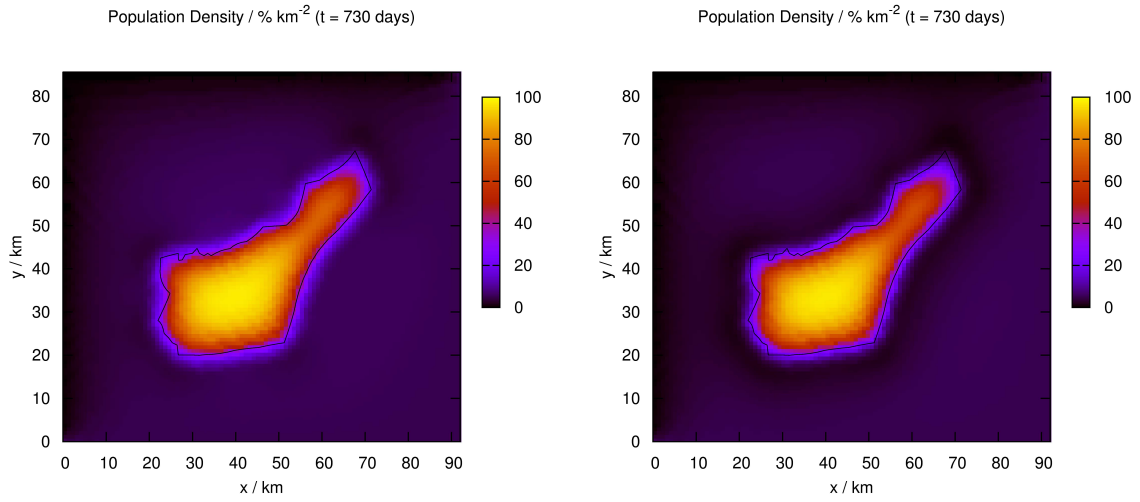


Figure 11: An approximately 5 km–wide barrier, after 2 years, for $\lambda = 0.08 \text{ km}^2 \text{ day}^{-1}$. At left, a $2 \% \text{ day}^{-1}$ mortality throughout the barrier. At right, a $4 \% \text{ day}^{-1}$ mortality throughout.

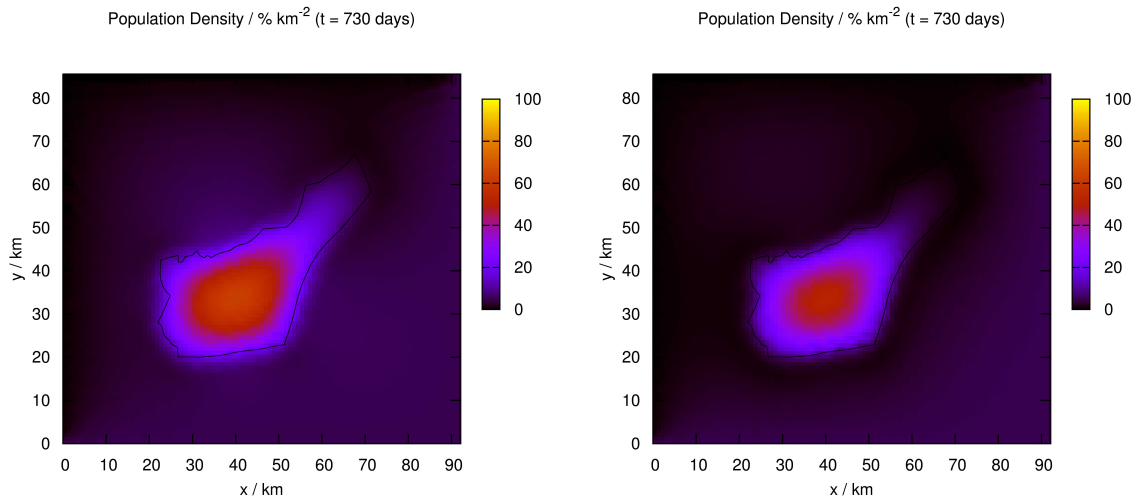


Figure 12: An approximately 5 km–wide barrier, after 2 years, for $\lambda = 0.32 \text{ km}^2 \text{ day}^{-1}$. At left, a $4 \% \text{ day}^{-1}$ mortality throughout the barrier. At right, a $12 \% \text{ day}^{-1}$ mortality. (At $8 \% \text{ day}^{-1}$ the barrier zone along the concave boundary was deemed to be not entirely vacant.)

8.4 Pour-Ons and the Subsequent Rebound, With or Without Suppression

8.4.1 $\lambda = 0.08 \text{ km}^2 \text{ day}^{-1}$

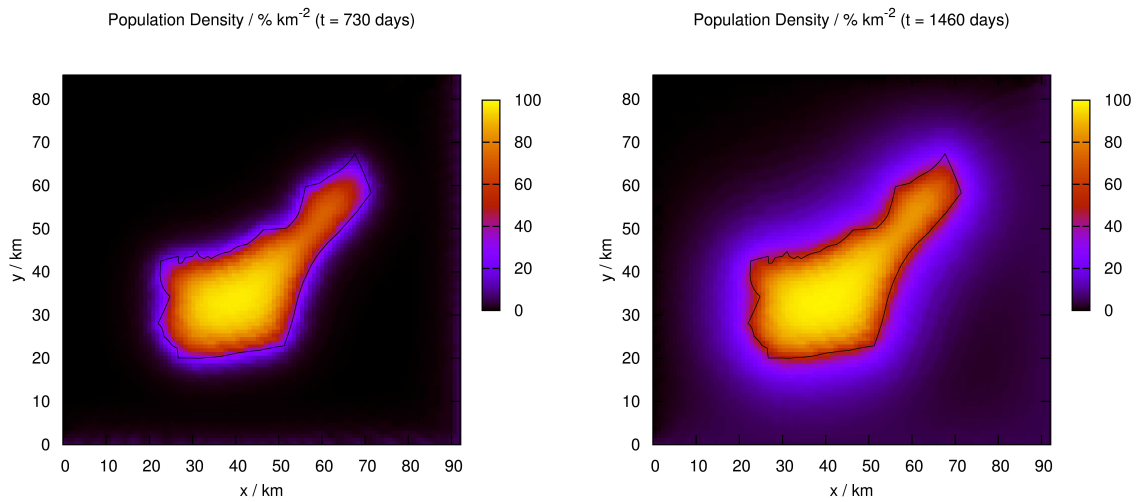


Figure 13: The result of a 2 % day⁻¹ mortality imposed everywhere outside the reserve for a period of 2 years (left); the rebound after a further 2 years (right).

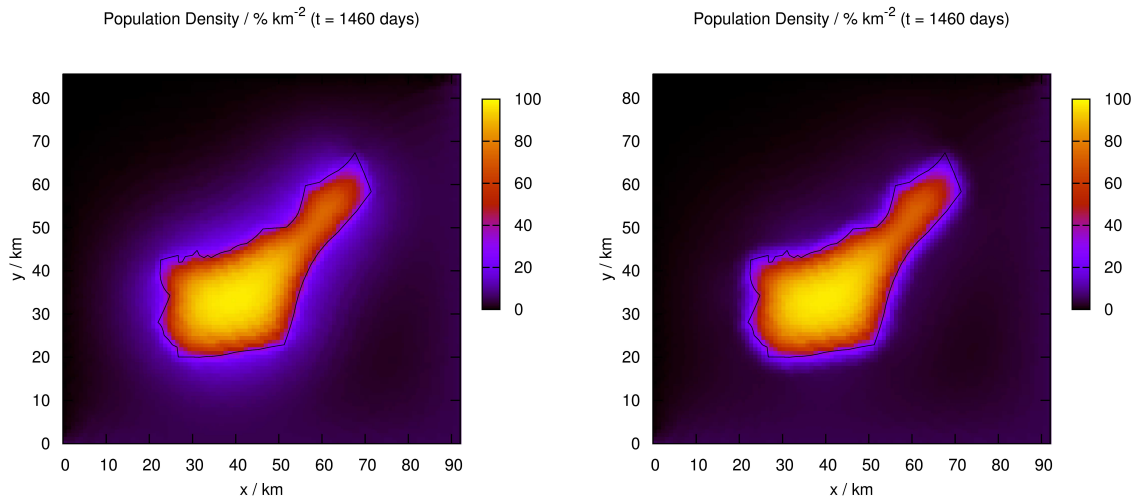


Figure 14: The rebound suppressed by a barrier with a mortality of 2 % day⁻¹ everywhere inside it. At left, an approximately 2.5 km-wide barrier; At right, an approximately 5 km-wide barrier.

8.4.2 $\lambda = 0.32 \text{ km}^2 \text{ day}^{-1}$

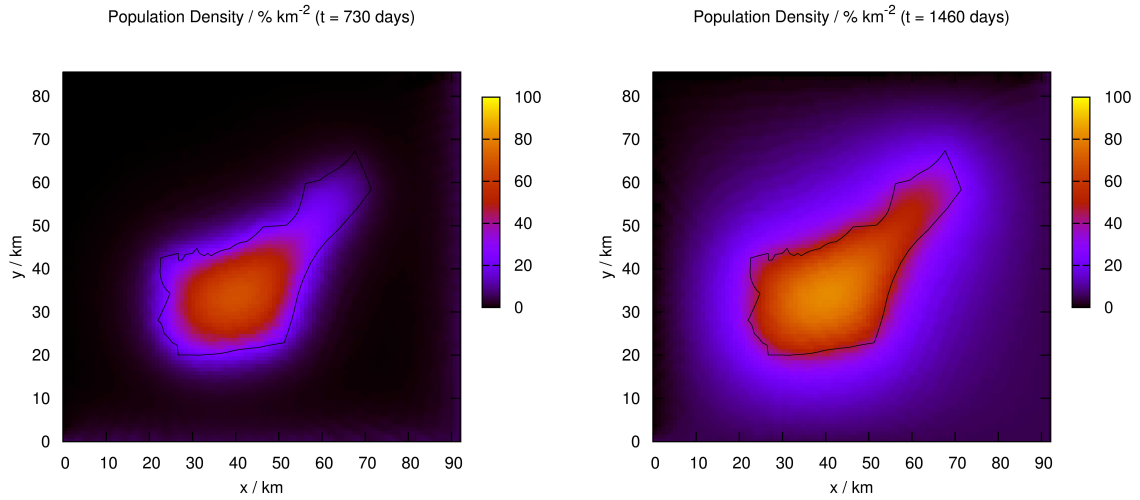


Figure 15: The result of a 2 % day⁻¹ mortality imposed everywhere outside the reserve for a period of 2 years (left); the rebound after a further 2 years (right).

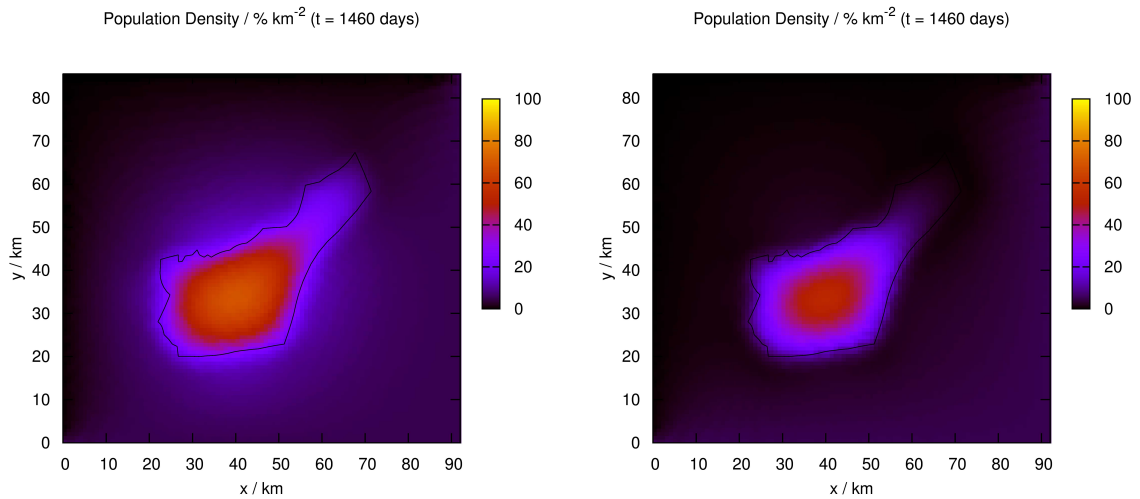


Figure 16: The rebound suppressed by an approximately 5 km-wide barrier. At left, a 2 % day⁻¹ mortality throughout the barrier. At right, an 8 % day⁻¹ mortality throughout the barrier.

8.5 The Implications of the Diffusion Coefficient for Eradication and Any Subsequent Rebound

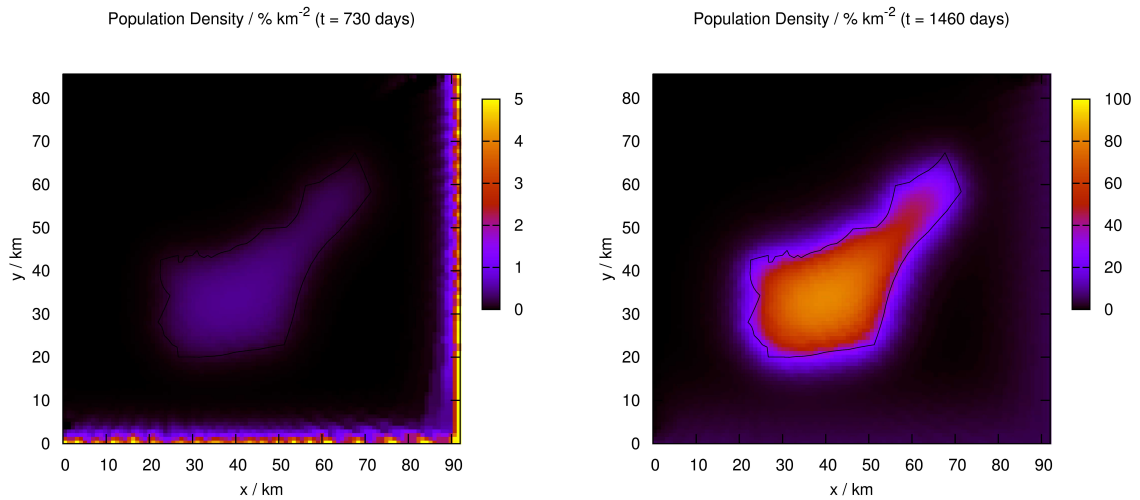


Figure 17: $\lambda = 0.04 \text{ km}^2 \text{ day}^{-1}$. The result of a $2 \% \text{ day}^{-1}$ mortality imposed everywhere for a period of 2 years (left); the population rebound after a further two years (right).

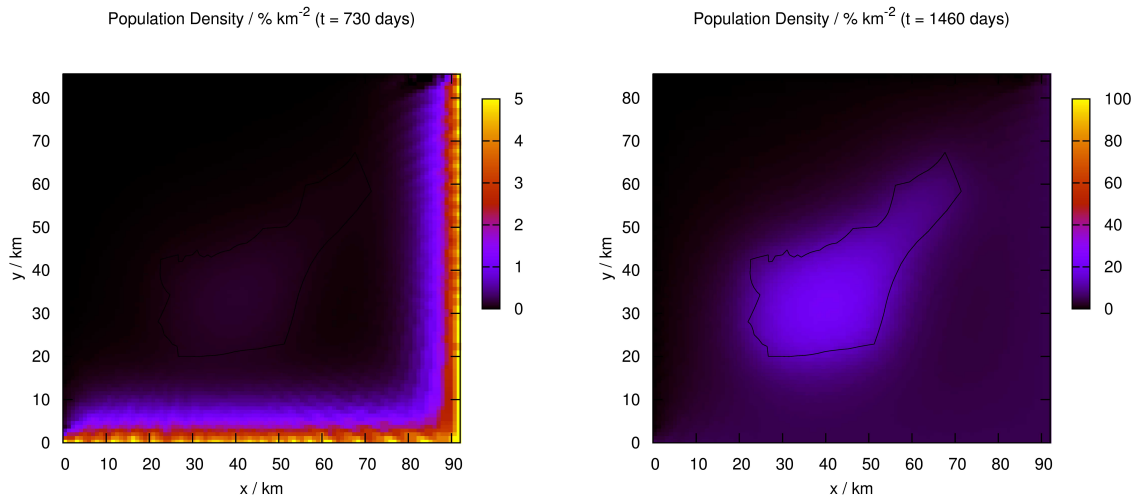


Figure 18: $\lambda = 0.32 \text{ km}^2 \text{ day}^{-1}$. The result of a $2 \% \text{ day}^{-1}$ mortality imposed everywhere for a period of 2 years (left); the population rebound after a further two years (right).

8.6 ‘Pumping’ Out the Reserve Population from the Boundary

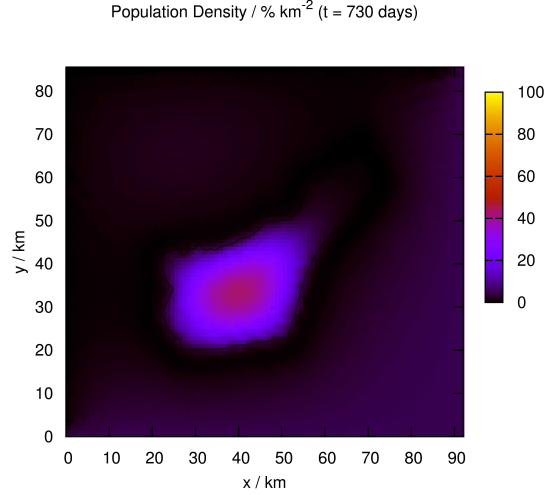


Figure 19: The result of a 5 km–wide barrier in which a 50 % day⁻¹ mortality is imposed for a period of 2 years ($\lambda = 0.32 \text{ km}^2 \text{ day}^{-1}$).

9 Improving Barrier Mortality Rates

Odour–baited targets and cattle treated with so–called ‘pour–ons’ are the means by which tsetse barriers can be constructed.

9.1 Translating Barrier Mortality into Odour–Baited Targets and the Possible Revelation of Competition

The definitive experimental work involving target barriers for *G. austeni* and *G. brevipalpis* is that of ESTERHUIZEN, KAPPMEIER GREEN, NEVILL and VAN DEN BOSSCHE [7]. Essentially they barricaded a small peninsula with targets. They also placed targets on the peninsula itself and measured the decline of *G. austeni* and *G. brevipalpis* in relation to the target density on the peninsula. At a target density of 8 km^{-2} *G. austeni* was found to decline at a rate of around -0.014 day^{-1} .

Despite good estimates of the rate of *G. austeni* decline, choosing other parameters in WILLIAMS, DRANSFIELD and BRIGHTWELL [32]’s seminal equation is something of a heuristic exercise. If one takes cognizance of ROGERS and RANDOLPH [26]’s findings on predation, the pupal water loss model of CHILDS [4] etc., a 2 % day⁻¹ pupal mortality rate would certainly not be unreasonable for the species in question. Natural mortality for the nulliparous cohort is known

to be high and, although it does not fall victim to targets in the same proportions as adults, some still do (HARGROVE [14]). With this fact in mind the nulliparous stage flies were assigned a natural mortality of $2.2\% \text{ day}^{-1}$ and assumed to have a target-related mortality one half that of adults. At 22.1°C the relevant formulae¹ for the first and subsequent interlarval periods predict 17.5 days and 10.5 days respectively. The formula 6 gives a puparial duration of 34.6 days for females. A miscarriage rate of 5 %, and therefore a fecundity of 0.475 was used (in keeping with WILLIAMS, DRANSFIELD and BRIGHTWELL [32]).

Solving WILLIAMS, DRANSFIELD and BRIGHTWELL [32]'s equation using the aforementioned parameters and Newton's method suggested the 8 km^{-2} target density of ESTERHUIZEN, KAPPMEIER GREEN, NEVILL and VAN DEN BOSSCHE [7] was equivalent to an artificially imposed mortality of $2.39\% \text{ day}^{-1}$ ($0.30\% \text{ day}^{-1}$ per target). Target-related mortality is obviously much lower for these forest species. (By comparison, a single odour-baited target¹ kills $2\% \text{ day}^{-1}$ of the female *G. pallidipes* population HARGROVE [11].)

So far as *G. brevipalpis* is concerned, ESTERHUIZEN, KAPPMEIER GREEN, NEVILL and VAN DEN BOSSCHE [7]'s results are not as clear. Indeed, the results of this work suggest their barrier-zone might have been completely ineffectual against a very mobile *G. brevipalpis*. Another possibility is that *G. brevipalpis* is completely impartial to odour-baited targets. Yet something certainly did happen in both target-containing sectors when the concentration of targets reached a density of 8 km^{-2} . *G. brevipalpis* initially declined at a rate indicative of an imposed mortality of $0.63\% \text{ day}^{-1}$ (again, using WILLIAMS, DRANSFIELD and BRIGHTWELL [32]'s esteemed equation). A subsequent reversal of this decline then coincided with the demise of *G. austeni* and the *G. brevipalpis* population grew to levels never previously attained; in spite of the targets. A number of explanations spring to mind. One argument is that the data is too poor, that what is being observed is simply random noise, should be ignored. Another possibility is that there was a delay in recolonization by this highly mobile species. Certainly one has good reason to suspect an element of diffusion to be operative, even if not the overriding analysis. Why then, the delay? There could be reasons.

An alternative explanation is that the reversal in fortune of the one species coincided with the demise of the other due to the two being in competition: So deleterious was the presence of *G. austeni* to *G. brevipalpis* that its removal is able to counteract the imposition of a $0.63\% \text{ day}^{-1}$ mortality on *G. brevipalpis* (a decline rate of -0.0039 day^{-1}). In retrospect, such a situation might have been anticipated. Indeed, one of the posits of this model is that the limitations on growth at pupal sites are density dependent. Pupal habitat for *G. brevipalpis* is more stringently confined than for *G. austeni* (according to demonstrations of the pupal water loss model in CHILDS [4]) and the *G. austeni* puparial duration is a full 20 % shorter than that of *G. brevipalpis* (PARKER [22]). One would imagine *G. brevipalpis* also has an adverse effect on *G. austeni*. Just how severe and whether or not it can be exploited, is not evident. Further experimentation is required. That ESTERHUIZEN, KAPPMEIER GREEN, NEVILL and VAN DEN BOSSCHE [7] were simply not able to measure a true value for the target-related mortality of *G. brevipalpis*, owing to high diffusion rates, is their own conclusion.

¹HARGROVE [13]'s improved EAST AFRICAN HIGH COMMISSION [1] formulae.

¹as specified in VALE, HARGROVE, COCKBILL and PHELPS [30]

If the accepted wisdom that the effect of uniformly distributed targets is additive, then a given mortality may be designed in terms of Table 3 as follows.

δ / day^{-1}	0.02	0.04	0.08	0.12
<i>G. austeni</i>	7	13	27	40
<i>G. brevipalpis</i>	25	51	102	152
<i>G. pallidipes</i>	1	2	4	6

Table 3: The number of targets per km^2 which will produce a required daily mortality, δ , for each species.

9.2 Tethered, Treated Cattle

Unpublished experiments by S. J. Torr (reported in HARGROVE, TORR and KINDNESS [16]) suggest that a single odour-baited target kills the equivalent number of *G. pallidipes* females in 1 km^2 as an insecticide-treated ox of weight 400kg does in a day. Since ESTERHUIZEN, KAPPMEIER GREEN, NEVILL and VAN DEN BOSSCHE [7] used the same $1.5 \times 1 \text{ m}$, black-blue-black targets (manufactured by Bonar Industries, Harare)¹, the corresponding target-related mortality should apply to the ox for *G. austeni* and *G. brevipalpis*; assuming these species do not discriminate any differently between the chemical signatures of the beast and the target.

9.3 Treated Herds

In HARGROVE, HOLLOWAY, VALE, GOUGH and HALL [15] it was determined that tsetse catches changed with the tonnage of cattle, m , in a ventilated shed and could be described by

$$\delta \propto 4m^{0.475}.$$

Torr's experiment (reported in HARGROVE, TORR and KINDNESS [16]) allows the constants in the simplistic model,

$$\begin{bmatrix} 0.0030 \\ 0.00079 \\ 0.02 \end{bmatrix} = 4 \begin{bmatrix} c_{austeni} \\ c_{brevipalpis} \\ c_{pallidipes} \end{bmatrix} 0.4^{0.475},$$

¹ESTERHUIZEN [6] and HARGROVE [14]

to be determined. The minimum tonnage of cattle required to induce a given daily mortality in a square kilometre is therefore given by Table 4.

species	<i>G. austeni</i>	<i>G. brevipalpis</i>	<i>G. pallidipes</i>
herd mass / tons km ⁻²	$0.4 \left(\frac{\delta}{0.0030} \right)^{\frac{1}{0.475}}$	$0.4 \left(\frac{\delta}{0.00079} \right)^{\frac{1}{0.475}}$	$0.4 \left(\frac{\delta}{0.02} \right)^{\frac{1}{0.475}}$

Table 4: The treated herd mass required to bring about a given mortality, δ , in each species.

10 Conclusions

The premise that the entire reserve, and it alone, is a problem is not as valid for *G. austeni* as it is in the case of *G. brevipalpis* (Figures 7 and 4). In the case of *G. austeni* it may well be worth singling out individual locii for barricading (e.g. the Hluhluwe Dam, its backwater and the area in the vicinity of the confluence of the Black iMfolozi and White iMfolozi rivers), based on CHILDS [4] and HENDRICKX [17].

The diffusion rate of *G. austeni* is probably around 0.04 km² day⁻¹, if the reserve exerts an influence on surrounding population levels 7 to 10 km from its boundaries. If, however, the influence is as great as 10 to 15 km, the diffusion rate is as high as 0.08 km² day⁻¹ (Figures 5 and 7). The diffusion rate of *G. brevipalpis* can be assumed to be around 0.32 km² day⁻¹, if the reserve exerts an influence on surrounding population levels as distant as 20 to 25 km from its boundaries. If, however, this influence is as little as 15 to 20 km, the diffusion rate might be as low as 0.16 km² day⁻¹ (Figures 4 and 6).

Based on the worst-case values in terms of which the problem was phrased, the simulations suggest that the temporary imposition of a 2 % day⁻¹ mortality everywhere outside the reserve for a period of 2 years will have no lasting effect on the influence of the reserve when it comes to either population; although it certainly will eradicate tsetse from areas of poor habitat, outside the reserve (Figures 13 to 16). It is doubtful whether the populations within the reserve can be ‘siphoned’ or ‘pumped out’ to extinction, or even the 20% level, from outside the reserve boundary (Figure 19).

The influence of the reserve on surrounding *G. austeni* population levels can be completely negated by a 5 km-wide barrier in which there is a mortality of 2 % day⁻¹ throughout (Figure 9). A 2.5 km-wide barrier of targets will, however, not suffice (Figure 8). For *G. brevipalpis* a 5 km-wide barrier to the same end will require a mortality of 4 % day⁻¹ throughout (Figure 12). Notice, however, that these measures are not in any way able to address the likelihood of more lethal strains and a higher level of trypanosome infection in flies close to the reserve

boundary, regardless of any reduction in their numbers.

A 5 km-wide barrier of odour-baited targets with a mortality of 4 % day⁻¹, throughout, should succeed in completely isolating a worst-case, Hluhluwe-iMfolozi *G. austeni* population and its associated more lethal strains and higher levels of trypanosomiasis from the surrounding areas (Figure 11). A more optimistic estimate of its mobility suggests a mortality of 2 % day⁻¹ might suffice (Figure 9). In the event that *G. brevipalpis* is shown to be a vector of trypanosomiasis, a worst-case mortality of 12 % day⁻¹, throughout, will be required to achieve the same end of complete isolation (Figure 12). A mortality of 8 % day⁻¹ can be used at non-concave boundaries. It is further recommended that any barrier include the surroundings of the Hluhluwe dam and its backwater, as if it were part of the reserve, based on CHILDS [4].

For a given mortality, more mobile species are found to be more vulnerable to eradication than more sedentary species while the opposite is true for containment. The scenarios depicted in Figures 17 and 18 demonstrate, firstly, that high diffusion rates are more amenable to eradication, since the same local consistency is not required. Species with high diffusion rates are vulnerable to controls which are geographically more remote. Secondly, high diffusion rates lead to a much weaker recovery from levels close to extinction¹. The reason is that there is a tendency to disperse which is not efficacious at the lower levels of logistic growth. This might seem obvious to the reader, yet one popular theory for the phenomenal Lambwe Valley rebound (reported in TURNER AND BRIGHTWELL [28] and HARGROVE [12]) is re-invasion, usually quoting the high mobility of *G. pallidipes*. A very large neighbouring population would be required for such a rebound to be attributed to re-invasion. More likely explanations would be the over-estimation of the temperature at pupal sites, or a problem with insecticide application.

Susceptability to odour-baited targets and existing population distributions aside, one is now presented with a scenario in which *G. brevipalpis* may be more vulnerable to eradication than containment and vice versa for *G. austeni*. Yet whether or not *G. brevipalpis* is even an agent of infection is still a moot point (MOTLOANG, MASUMU, VAN DEN BOSSCHE, MAJIWA and LATIF [20]). *G. austeni*, in contrast, is without the slightest doubt a highly competent vector of trypanosomiasis. The possibility that eliminating *G. brevipalpis* will create further opportunity for *G. austeni* and, consequently, trypanosomiasis needs to be considered. The experimental results of ESTERHUIZEN, KAPPMEIER GREEN, NEVILL and VAN DEN BOSSCHE [7] can be interpreted to lend credence to exactly such a theory. They could suggest intense competition between the two species, to the extent that *G. brevipalpis* may actually benefit from odour-baited targets should their density be sufficient to eliminate *G. austeni* only. The existence of a reciprocal effect on *G. austeni* may be well worth investigating. Then again, what is observed could simply be a delayed invasion response or even random noise. That ESTERHUIZEN, KAPPMEIER GREEN, NEVILL and VAN DEN BOSSCHE [7] were simply not able to measure the target-related mortality of *G. brevipalpis*, owing to high diffusion rates, is an alternative conclusion.

The required density of treated herds is not practical for the purposes of barriers and containment. While tethered, deltamethrin-treated cattle can be used as an alternative to odour-baited

¹Although this could be an artefact of assuming more mobile species have a growth rate the same as more sedentary species.

targets for savannah species, the substitution is impractical in the case of forest species. (Individually tethered, treated cattle distributed uniformly throughout a barrier zone may be less likely than targets to fall victim to the tragedy-of-commons type mentality known to prevail among the local population.)

The K. P. P. equation can be solved by way of the application of the finite element method for the spatial discretisation and a backward difference for the temporal discretisation. This strategy in combination with the linearisation and iteration of the nonlinear term also worked well for Fisher's equation, with good convergence for the range of conditions investigated.

A combination of warm temperatures, low imposed mortalities and long two year cycles gave the population ample time to re-equilibrate with the result that there was no discernable difference in the results obtained from either model. The reader should, nonetheless, be cautioned against the use of a model based on an unmodified Fisher's equation when faced with rather more severe circumstances e.g. the catastrophic mortalities imposed by aerial spraying and lower temperatures. Attributing subsequent growth to a current, as opposed to historical, population would be profoundly incorrect under such circumstances. Under the same circumstances, the model based on historical parentage would not take the subsequent reproductive phase entrainment and altered age profile into account. Reproductive rates would initially be over-estimated, later, under-estimated and so on. Unlike Fisher's equation, however, the model is expected to recover.

What of the danger of an altered age profile in the light of a longer than usual first interlarval period? Comparison of the results of the two models suggests that if circumstances allow the population to re-equilibrate there are unlikely to be any problems.

10.1 Recommendations to Management

In the case of *G. austeni* a permanent, 5 km-wide isolating barrier should be maintained on the Eastern side of the reserve, around the Hluhluwe Dam and its backwater, the vicinity of the confluence of the Black iMfolozi and White iMfolozi rivers as well as other locii outside the reserve. The required mortality of the barrier should be no less than 4 % day⁻¹ for the worst-case scenario. This value corresponds to a deployment of odour-baited targets¹ with a minimum density of 13 km⁻². If, however, *G. austeni* is only diffusing at a rate as low as 0.04 km² day⁻¹, then the required number of odour-baited targets¹ is 7 km⁻². Tethered, treated cattle can also be used as substitutes. Periodically rotating them in and out of the barrier zone would prevent a loss of resistance to tick-bourne diseases and an enzootic condition.

That ESTERHUIZEN, KAPPMEIER GREEN, NEVILL and VAN DEN BOSSCHE [7] were simply not able to measure the target-related mortality of *G. brevipalpis*, owing to high diffusion rates, is one conclusion. Another is that the two species may be in competition, a possibility which may or may not be exploitable. The impartiality of *G. brevipalpis* to odour-baited targets is, in any event, obviously a concern. Should this species be conclusively shown to be a vector, a

¹As used by ESTERHUIZEN, KAPPMEIER GREEN, NEVILL and VAN DEN BOSSCHE [7]

permanent, 5 km-wide barrier around the reserve as well as that part of the Hluhluwe dam and its backwater lying outside the reserve would need to be maintained. The required mortality of the barrier could be as high as $12\% \text{ day}^{-1\dagger}$ to achieve complete isolation. This is obviously not practical in terms of what one can only surmise is the mortality of current odour-baited target technology. The less ambitious goal of negating the reserves influence on the surrounding *G. brevipalpis* population would require a mortality of $4\% \text{ day}^{-1}$, throughout. This value corresponds to a deployment of odour-baited targets with a minimum density of 51 km^{-2} ; again a clearly impractical proposition.

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This work is obviously a synthesis of the monumental research efforts and pioneering work carried out by the likes of Jackson, Bursell, Phelps, Vale, Rogers, Williams and Hargrove, to name but a few. This work is, to a large extent, theirs with the appropriate numerical methods applied.

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[†] $12\% \text{ day}^{-1}$ where the boundary is concave and $8\% \text{ day}^{-1}$ where convex

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Addendum

The Change in Population Density Due to Migration

Consider the hypothetical scenario of a mobile population in the absence of either reproduction or mortality (external and artificially imposed, or otherwise). Let $\Omega(t)$ be an arbitrary sub-

volume of flies with boundary $\Gamma(t)$. Then biomass should be conserved so that

$$\begin{aligned} \frac{D}{Dt} \int_{\Omega(t)} \rho \, d\Omega &= 0 && \text{(rate of change of mass with time = 0)} \\ \frac{d}{dt} \int_{\Omega_0} \rho_0 J_0 \, d\Omega_0 &= 0 && \text{(reformulating in the material} \\ &&& \text{configuration, } \Omega_0) \\ \int_{\Omega_0} \frac{d}{dt} \{ \rho_0 J_0 \} \, d\Omega_0 &= 0 && \text{(since limits are not time dependent} \\ &&& \text{in the material configuration)} \\ \int_{\Omega_0} \left(\rho_0 \dot{J}_0 + \dot{\rho}_0 J_0 \right) \, d\Omega_0 &= 0 && \text{(by the product rule)} \\ \int_{\Omega(t)} (\dot{\rho} + \rho \operatorname{div} \mathbf{v}) \, d\Omega &= 0 && \text{(using } \dot{J}_0 = J_0 \operatorname{div} \mathbf{v})^\dagger, \end{aligned}$$

in which \mathbf{v} is velocity. Since the volume was arbitrary it follows that the integrand must be zero. That is

$$\dot{\rho} + \rho \operatorname{div} \mathbf{v} = 0.$$

Now consider this biomass conservation in the context of another arbitrary sub-volume, this time of habitat, Ω_h , with boundary Γ_h .

$$\begin{aligned} \int_{\Omega_h} \left(\frac{\partial \rho}{\partial t} + \nabla \rho \cdot \mathbf{v} + \rho \operatorname{div} \mathbf{v} \right) \, d\Omega_h &= 0 && \text{(expanding } \dot{\rho}) \\ \int_{\Omega_h} \left(\frac{\partial \rho}{\partial t} + \operatorname{div} \{ \rho \mathbf{v} \} \right) \, d\Omega_h &= 0 && \text{(by the product rule)} \\ \int_{\Omega_h} \frac{\partial \rho}{\partial t} \, dV + \int_{\Gamma_h} \rho \mathbf{v} \cdot \mathbf{n} \, d\Gamma_h &= 0 && \text{(by the divergence theorem)} \\ \int_{\Omega_h} \frac{\partial \rho}{\partial t} \, dV - \int_{\Gamma_h} -\lambda \nabla \rho \cdot (-\mathbf{n}) \, d\Gamma_h &= 0 && \text{(by Fick's 1st law)} \\ \int_{\Omega_h} \left(\frac{\partial \rho}{\partial t} - \lambda \operatorname{div} \nabla \rho \right) \, d\Omega_h &= 0 && \text{(by the divergence theorem).} \end{aligned}$$

Since the volume was arbitrary it again follows that the integrand must be zero. That is

$$\frac{\partial \rho}{\partial t} = \lambda \operatorname{div} \nabla \rho,$$

in which λ is the diffusion coefficient.

[†]The material derivative of the Jacobian is given by the kinematic relation $\dot{J}_0 = J_0 \operatorname{div} \mathbf{v}$, a result demonstrated in most popular textbooks on continuum mechanics (eg. MARSDEN and HUGHES [19]).